

Comparative Assessment of Salivary Dopamine and Acetylcholinesterase Levels in Smokers and Non-Smokers

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Abstract Background: Nicotine, the principal psychoactive compound in tobacco, alters key neurochemical pathways, particularly those involving dopamine and acetylcholinesterase (AChE). These neurotransmitter changes may underlie the development of nicotine dependence and its cognitive and behavioral consequences. Comparing these biomarkers in smokers and non-smokers provides insight into the neurophysiological impact of chronic tobacco use. **Objective:** To assess and compare salivary dopamine levels and acetylcholinesterase (AChE) activity in smokers and non-smokers. **Methods:** An *in vivo* cross-sectional study was conducted among 240 healthy male participants aged 30 to 35 years, comprising 120 smokers and 120 non-smokers. Salivary levels of dopamine and AChE were measured using enzyme-linked immunosorbent assay (ELISA). Due to non-normal data distribution, the Mann-Whitney U test was used for statistical analysis, with significance set at $p < 0.05$. **Results:** Smokers demonstrated significantly higher AChE activity (mean: 126.38 nmol/l) compared to non-smokers (mean: 112.9 nmol/L; $p < 0.001$), suggesting altered cholinergic function. Dopamine levels were also significantly elevated in smokers (mean: 29.84 ng/ml) versus non-smokers (mean: 23.83 ng/ml; $p = 0.002$), indicating stimulation of the dopaminergic system likely linked to nicotine exposure. These results underscore the biochemical influence of chronic smoking on neural regulation and addiction pathways. **Conclusion:** The study found elevated salivary dopamine and AChE levels in smokers, indicating that nicotine use may contribute to both dopaminergic overactivation and cholinergic dysregulation. These neurochemical changes may reinforce addiction and impair cognitive processes. The findings support the need for neurobiologically informed smoking cessation strategies. Further research should investigate whether these biochemical alterations are reversible following smoking cessation.

Key Words Acetylcholinesterase (AChE), Dopamine, Neurotransmitters, Cognitive Function, Cholinergic System, Dopaminergic System, Smoking Cessation

INTRODUCTION

Tobacco use remains a major global health concern, contributing to a wide range of systemic and neurological disorders. Nicotine, the primary psychoactive compound in tobacco, is a potent stimulant that affects various neurotransmitter systems, particularly dopamine and acetylcholine, both of which are involved in cognition, mood regulation and addiction pathways [1]. While nicotine itself is not directly carcinogenic, its ability to reinforce smoking behavior results in prolonged exposure to numerous carcinogens found in tobacco products.

Though often perceived as a coping mechanism for stress, chronic smoking exacerbates psychological stress and

dysregulates neurochemical systems over time [2]. This dysregulation significantly impairs cognitive flexibility, emotional stability and decision-making. Smoking is habitually integrated into daily routines, often becoming a deeply ingrained behavior. Despite its short-term perceived relief, the long-term consequences include elevated risks for chronic respiratory and cardiovascular diseases, periodontal deterioration and delayed wound healing [3,4].

Nicotine's structural similarity to acetylcholine (ACh) allows it to bind to nicotinic acetylcholine receptors (nAChRs), mimicking ACh's effects and promoting neurotransmitter release. This interaction facilitates heightened arousal and cognitive engagement but also

contributes to addiction and eventual neurochemical imbalance [5]. Through the mesolimbic dopaminergic system, nicotine stimulates dopamine release in the nucleus accumbens, reinforcing pleasure-seeking behaviors. Chronic exposure to nicotine leads to desensitization and upregulation of nAChRs, further entrenching nicotine dependence. Additionally, nicotine influences other neurotransmitters such as serotonin, gamma-aminobutyric acid (GABA), glutamate and norepinephrine, which are critical for mood regulation and memory [6]. These interactions contribute to withdrawal symptoms and increased relapse rates, making cessation difficult.

Over time, chronic smoking initiates oxidative stress and neuroinflammation, contributing to structural brain changes, impaired synaptic plasticity and increased susceptibility to neurodegenerative disorders [7]. This biochemical complexity underscores the need for investigating specific neurochemical alterations linked to nicotine exposure.

Acetylcholine plays a vital role in neural signaling, memory formation and muscle activation. Its levels are tightly regulated by acetylcholinesterase (AChE), an enzyme that terminates cholinergic signaling by breaking down ACh in the synaptic cleft. AChE not only facilitates neurotransmission but also contributes to neuronal development and synaptic remodeling [8]. Research indicates that chronic nicotine exposure may increase AChE activity, thereby disrupting cholinergic balance and impairing cognitive processes such as attention and memory in smokers [9].

Dopamine, a key neurotransmitter in the brain's reward and motivation circuits, is directly stimulated by nicotine intake. Repeated exposure elevates dopamine levels, reinforcing smoking behavior and contributing to addiction. Over time, such stimulation leads to receptor desensitization, reduced transporter availability and dopaminergic dysfunction. These changes are associated with emotional disturbances, reduced motivation and an increased risk of dependence [10]. In non-smokers, dopamine regulation remains physiologically controlled, supporting balanced emotional and behavioral responses.

Excessive AChE activity has been associated with cognitive decline and neurodegenerative conditions such as Alzheimer's disease, raising concerns about the long-term neurological consequences of smoking [11]. Similarly, nicotine-induced modifications to dopaminergic signaling, including reduced receptor sensitivity and altered synaptic function, are thought to perpetuate addiction and increase the difficulty of cessation efforts [12].

Given these findings, there is a compelling need to examine the biochemical impact of smoking on neurotransmitter regulation. While numerous studies have explored nicotine's behavioral effects, limited data exist on its *in vivo* influence on both AChE activity and dopamine levels in human populations. This study aims to compare salivary dopamine and acetylcholinesterase concentrations in

smokers and non-smokers to better understand the biochemical mechanisms of nicotine dependence. These insights may contribute to improved strategies for smoking cessation and cognitive health preservation in chronic tobacco users.

METHODS

Participants and Study Design

This *in vivo* cross-sectional study was conducted among 240 healthy male volunteers aged 30 to 35 years, recruited from the local community in Thiruvallur district. The study population was divided equally into two groups: 120 current smokers and 120 non-smokers. Smokers were defined as individuals who had smoked at least 15 cigarettes per day for a minimum of 15 years. Non-smokers were age-matched participants who reported no history of smoking or use of any tobacco products. Participants with a history of central nervous system disorders, traumatic brain injury, or any psychiatric diagnosis as defined by the DSM-IV criteria were excluded. All participants underwent a physical examination to confirm their health status.

Biochemical Analysis Using ELISA

Saliva samples were collected from all participants to measure dopamine and acetylcholinesterase (AChE) levels. Enzyme-linked immunosorbent assay (ELISA) kits specific to rat AChE and human dopamine were used for analysis. The assays followed a two-site sandwich ELISA technique. In brief, microplates pre-coated with capture antibodies were incubated with standards and samples. Following incubation and washing, horseradish peroxidase (HRP)-conjugated detection antibodies were applied. After a subsequent wash, chromogen substrates were added, resulting in a color change proportional to the analyte concentration. The reaction was stopped with a stop solution and Optical Density (OD) was measured at 450 nm using a microplate reader. The procedure followed the manufacturer's protocol [13].

Statistical Analysis

Data were tested for normality using the Kolmogorov-Smirnov and Shapiro-Wilk tests. Both tests indicated non-normal distribution ($p < 0.05$) for dopamine and AChE levels across all groups. As a result, non-parametric statistical methods were applied. Group comparisons were conducted using the Mann-Whitney U-test. Statistical analysis was performed using IBM SPSS Statistics (version XX) and a p-value of less than 0.05 was considered statistically significant.

Ethical Considerations

The study protocol was reviewed and approved by the Institutional Ethics Committee of Saveetha Institute of Medical and Technical Sciences. Written informed consent was obtained from all participants before enrollment.

RESULTS

The study involved 240 male participants between the ages of 30 and 35, with an equal division of 120 smokers and 120 non-smokers. All participants were long-term residents of the Thiruvallur district and were selected based on specific criteria to ensure they had no prior history of neurological disorders, psychiatric conditions, or head trauma.

The average age among smokers was 31 years ($SD = 3.4$), while non-smokers had a slightly higher mean age of 32 years ($SD = 4.1$), with no significant statistical difference between the two groups ($p > 0.05$). The body mass index (BMI) was comparable, with smokers having a mean BMI of 24.8 ($SD = 1.9$) and non-smokers averaging 24.0 ($SD = 2.6$), both within the normal range and showing no notable variation ($p > 0.05$).

The smoking group had a history of consuming tobacco daily for a minimum of 15 years, with an average of 20 cigarettes per day, ranging from 16 to 25 cigarettes. In contrast, non-smokers had not engaged in regular smoking (Figure 1). To ensure reliable comparisons, the study maintained demographic uniformity between the groups, reducing potential confounding influences so that any observed biochemical differences could be linked primarily to smoking habits rather than variations in baseline characteristics.

The Mann-Whitney U-test revealed a statistically significant difference in OD and nmol/l distributions between the groups ($p < 0.001$), leading to the rejection of the null hypothesis. These findings suggest that OD and nmol/l levels vary considerably among the studied groups. As the study population primarily comprised individuals aged 30 to 35 years, age-related variations are unlikely to be a major confounding factor. Instead, the observed differences

may be influenced by underlying biological, environmental, or clinical factors. These results emphasize the need for further research to identify the determinants of OD and nmol/l variations, which may have implications for disease pathophysiology, biomarker evaluation, or therapeutic strategies.

The results of this study indicate a significant difference in acetylcholine esterase and dopamine levels between smokers and non-smokers. Smokers had higher acetylcholine esterase levels (126 nmol/l) compared to non-smokers (112.9 nmol/l), with a statistically significant difference ($p < 0.001$). This suggests that smoking may upregulate acetylcholine esterase activity, potentially affecting cholinergic neurotransmission. Since acetylcholine is involved in cognitive functions, increased enzyme activity may lead to its faster breakdown, impacting memory, attention and neural processing in smokers. This aligns with prior studies indicating that nicotine exposure alters cholinergic pathways, potentially contributing to both cognitive stimulation and long-term neural adaptation (Table 1, 2).

Similarly, the dopamine levels were also significantly higher in smokers (29.84 ng/ml) than in non-smokers (23.83 ng/ml), with a p-value of 0.002. Dopamine plays a crucial role in the brain's reward system and its increase in smokers suggests that nicotine stimulates dopamine release, reinforcing addiction. This finding supports the well-documented role of nicotine in modulating dopamine pathways, contributing to dependence and withdrawal symptoms upon cessation. The higher variability in dopamine levels among smokers may indicate individual differences in nicotine metabolism and sensitivity to its rewarding effects (Table 3, 4).

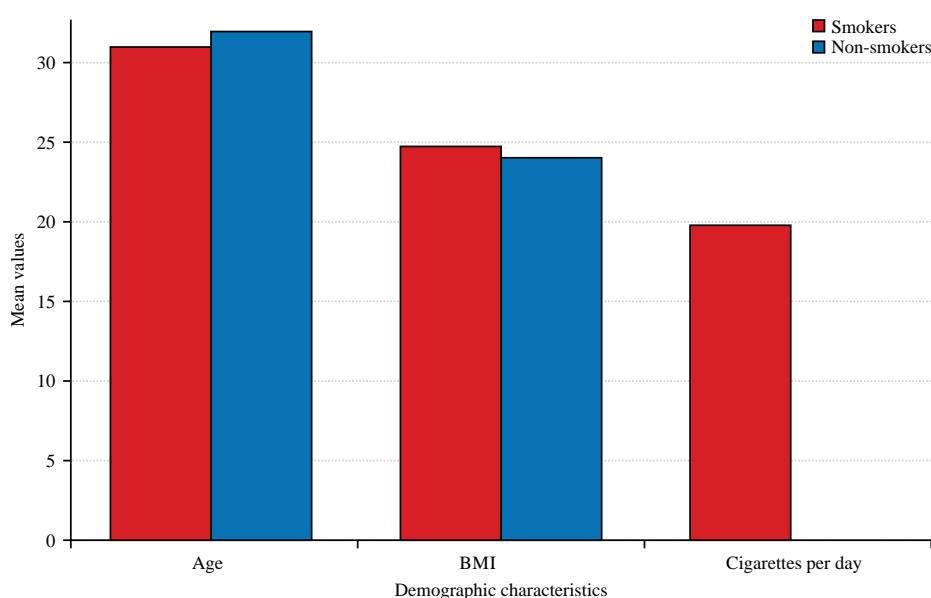


Figure 1: Comparison of demographic characteristics between smokers and non smokers

Table 1: Acetylcholinesterase level in smokers and non smokers

Salivary Acetylcholine esterase (nmol/l)				
	N	Mean	Standard deviation	Standard error Mean
Smokers	120	126.38	13.66	1.24
Non smokers	120	112.90	12.7	1.16

Table 2: Mann-Whitney U test for the acetylcholinesterase level in smokers and non smokers

Salivary Acetylcholine esterase (nmol/l)					
	N	Mean rank	U	Z	P
Smokers	120	156.11	2927	-7.998	0.000
Non smokers	120	84.89			

Table3: Dopamine level in smokers and non smokers

Salivary Dopamine (ng/ml)				
	N	Mean	Standard deviation	Standard error Mean
Smokers	120	29.84	7.52	0.68
Non smokers	120	28.33	8.5	0.78

Table 4: Mann-Whitney U test for the dopamine level in smokers and non smokers

Salivary Acetylcholine esterase (nmol/l)					
	N	Mean rank	U	Z	P
Smokers	120	156.11	2917	-7.998	0.002
Non smokers	120	84.89			

DISCUSSION

This study provides key insights into the neurochemical alterations associated with chronic smoking, particularly its effects on acetylcholinesterase (AChE) activity and dopamine levels. These findings contribute to a better understanding of the biochemical mechanisms underlying nicotine dependence and its potential impact on cognitive and emotional regulation. Elevated AChE activity and increased dopamine levels observed in smokers point to significant neuromodulatory effects induced by long-term nicotine exposure, supporting the need for integrated intervention strategies that address both addiction and its neurobiological consequences.

Acetylcholinesterase plays a vital role in regulating acetylcholine at synaptic junctions, thereby ensuring balanced cholinergic transmission. The current results demonstrate that smokers consistently exhibit higher AChE activity compared to non-smokers. This upregulation may disturb cholinergic homeostasis, leading to reduced acetylcholine availability and impaired neurotransmission. As acetylcholine is crucial for learning and memory, this disruption may underlie cognitive deficits often reported among chronic smokers. These observations align with previous studies suggesting nicotine-induced enhancement of AChE activity and associated neurochemical imbalances [14].

Dopamine, central to the brain's reward system, is strongly modulated by nicotine. The elevated dopamine levels observed in smokers in this study are consistent with nicotine's known effect of stimulating dopamine release, particularly in mesolimbic areas. This dopaminergic surge

reinforces addictive behaviors and contributes to nicotine dependence [15]. Chronic exposure may also lead to long-term adaptations such as receptor desensitization and reduced receptor density, which can contribute to mood disturbances and a diminished ability to experience natural rewards [16].

While the study revealed significant differences between smokers and non-smokers in both AChE activity and dopamine levels, the variability within each group was relatively modest. This suggests that individual biological differences, including genetics, metabolism and smoking intensity, may also influence neurochemical responses to nicotine [17]. The reliability of these findings is supported by consistent calibration and Optical Density (OD) values during ELISA quantification, ensuring accuracy in biomarker assessment.

These results are in line with neuroimaging studies that report increased dopamine concentrations in key brain regions such as the putamen and caudate nuclei of the basal ganglia in smokers compared to non-smokers, with statistically significant differences [18]. Additional evidence indicates that striatal dopamine release is positively correlated with the level of nicotine dependence and is significantly greater in smokers following nicotine administration [19].

Slotkin's experimental work further supports these observations by showing that nicotine exposure upregulates AChE activity in the central nervous system, leading to disruption of cholinergic signaling. This biochemical imbalance has been associated with learning and memory impairments, reinforcing the link between chronic nicotine exposure and cognitive decline [20,21]. These findings align with the current study's results and highlight the need to consider AChE modulation in strategies aimed at mitigating neurocognitive dysfunction in smokers.

Furthermore, nicotine's dual modulation of both dopaminergic and cholinergic systems underscores its complex impact on brain chemistry. By enhancing dopamine release while altering AChE activity, nicotine creates a reinforcing feedback loop that sustains addiction while gradually impairing cognitive resilience. This dual impact suggests that recovery from nicotine dependence may require addressing both neurotransmitter systems simultaneously [22].

The alterations in AChE activity and dopamine levels observed in smokers underscore the importance of further research into the long-term effects of smoking on the nervous system. A deeper understanding of these neurochemical changes could inform the development of targeted pharmacological and behavioral interventions designed to restore cognitive function and emotional balance in individuals with nicotine dependence [23,24]. Moreover, investigating whether these changes are reversible after smoking cessation may offer valuable insights into neurobiological recovery and the broader health benefits of quitting tobacco.

CONCLUSION

This study demonstrates that chronic smoking is associated with significant alterations in key neurochemical markers, including elevated acetylcholinesterase activity and increased dopamine levels. These findings suggest that prolonged nicotine exposure disrupts both cholinergic and dopaminergic pathways, contributing to neurochemical imbalances that reinforce addiction and may impair cognitive function. The results underscore the neurobiological basis of nicotine dependence and highlight the potential risk for long-term cognitive and emotional disturbances in smokers. These insights may inform the development of targeted cessation strategies that address both the behavioral and biochemical dimensions of tobacco addiction. Further longitudinal research is warranted to evaluate the reversibility of these neurochemical changes following sustained smoking cessation.

Limitations

This study has certain limitations. The cross-sectional design does not allow for assessment of temporal changes in biomarker levels. A long-term follow-up would be necessary to evaluate how AChE activity and dopamine levels fluctuate over time, especially in relation to smoking cessation. Additionally, potential measurement errors in ELISA or inter-laboratory variability could influence the accuracy of biomarker quantification. The study also focused exclusively on male participants, limiting the generalizability of findings across genders.

Conflict of Interest

The authors declare no conflict of interest related to this study.

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